SUBARACHNOID SEROUS EXUDATION PRODUCTIVE OF PRESSURE SYMPTOMS, AFTER HEAD INJURIES.

Dr. G. L. Walton read a paper with the above title, and said that in cases offering moderate fibrile movement, prolongation of unconsciousness, and restlessness after trauma, it is of no vital moment whether we adopt the term contusion, bruising or laceration; but when to these symptoms local paralysis is added, the question of operation arises and exact ideas of the pathology become important. Operation over the area indicated sometimes shows only a tense dura, incision of which is followed by a gush of serum. Under the arachnoid membrane the greater part of the cortical cerebrospinal fluid is collected. Serum may be exuded into the subarachnoid space as well as transuded, and sometimes the swollen and congested brain found under the fluid on operation shows that we have to do with an exudation resulting from increased blood pressure, rather than with a compensatory process. Little attention has been paid to this process. The edema of Ballard accompanying hemorrhage is analogous; the serous meningitis of Quincke is analogous if not identical, in so far as he includes external meningitis, but he does not refer to this class of cases. The following cases were mentioned: A boy of six years was struck by a bicycle at noon one day, was restless and drowsy, and on the following day became unconscious, with unilateral paralysis including the face Operation was considered but postponed. The paralysis had disappeared within four days. A child, three and a half years old, fell from a swing, striking the head; was drowsy, and the next day one arm was paralyzed. The paralysis began to lessen on the third day, and rapidly disappeared. A young woman fell, striking the head violently. Unconsciousness, restlessness, vomiting, and hemiplegia appeared. Operation revealed a tense dura, incision of which was followed by a gush of serum. There was relief of pressure symptoms, but death ensued from the underlying condition. His conclusions were: 1. A severe blow may result in local bruising and congestion with subarachnoid serous exudation. 2. The fluid may be imprisoned and cause focal paralysis. 3. The process is not compensatory, and is allied to the serous meningitis of Quincke. 4. The lesion is self-limiting. 5. Diagnosis from hemorrhage is difficult. Atypical course, absence of steady increase of symptoms, and persistence of sensitiveness point to serous exudation. 6. Immediate operation is not necessarily indicated in focal paralysis, though, perhaps, always justifiable. 7. This condition is specially to be borne in mind before operating on children and young adults.

DISCUSSION.

Dr. Wm. N. Bullard—Two years or more ago, in a paper read before the Surgical Section of the Suffolk District Medical Society, and later printed in the Reports of the Boston City Hospital, I drew attention to the existence of increased intracranial and intradural pressure in cases of injury of the head. Further investigation and study of these cases has confirmed my belief that this condition is the pathological cause of many of the symptoms arising in these cases, and that the presence and degree of this increased intradural pressure is one of the most important factors in determining whether or not operation should be performed. I think that it may now be received as an accepted fact that in many cases, at least, of injuries of the head such an increase of intradural pressure exists. That this intradural pressure is due in part in some cases to effusion into the spaces between the meninges, and possibly into the tissues of the meninges themselves, is unquestionable. But it is yet undecided how far this effusion may be considered as the principal cause of the increase of intradural pressure, and how far a soaking of the brain tissue and a filling and enlargement of the intracranial vessels, especially those in the brain, contributes to this result. Certain it is that in many cases where, after injury, there is a marked intradural pressure, the opening of the dura does not let out any large amount of serous fluid.

As to how far the symptoms due to this increased pressure within the dura, when localized in the form of paralysis, hemiplegia or some other type, are to be considered as indications for surgical interference has not yet been fully determined. We know, however, that patients, who after injury to the head become gradually unconscious and shortly develop a hemiplegia, having, in short, such symptoms as would suggest a hemorrhage within the cranium, may recover spontaneously without treatment within a few hours. This is apparently more frequently the case in children than in adults. I would especially call attention in this relation to the case of Dr. Prince reported last winter.

Permit me, finally, to present my regrets at not being able to attend this meeting and to give my thanks to Dr. Walton for giving me a summary of his valuable paper. I believe this to be a most important subject, and that in its thorough knowledge and consideration lies the key to proper discrimination in operation in head injuries.

Dr. Morton Prince, of Boston—I think the theory of Dr. Walton is very plausible, but difficult of substantiation. I remember the case of a child who had a fall, and showed the classical symptoms of meningeal hemorrhage, such as unconsciousness following an interval of consciousness, hemiplegia, etc. Operation was deferred, and the child was perfectly well on the following day. The most plausible theory in this case is, I think, that of local edema.

Dr. B. Sachs, of New York—The theory advanced is not a new one. After head injuries three possibilities are to be considered: hemorrhage, local edema, and purulent meningitis Dr. Walton's view is natural and plausible. and I agree with his views as to differential diagnosis. I would advise deferring operation until there was positive proof of serious damage to the brain, at any rate until after the lapse of forty-eight hours or several days.

Dr. J. J. Putnam, of Boston—I assisted at an autopsy on a woman who, during convalescence from a severe influenza, had developed localized twitching of arm, leg and face every twenty minutes, followed twenty-four hours later by hemiplegia and rise of temperature. There were only edema and a wet, soft brain, and there was no local accumulation under the pia. I think the convulsions and hemiplegia were due to the general edema. The precise cause of the nerve irritation in these cases is not clear.

Dr. Hugh T. Patrick, of Chicago—I have had a case communicated to me almost exactly like that of Dr. Prince. A child received a fall, which was not severe, in the latter part of the afternoon, after which he played about as usual. At the supper table he suddenly let fall a fork from his right hand, and soon thereafter had a one-sided fit. Following this he became stuporous, and when seen, several hours later, was almost comatose; but a distinct hemiplegia was made out. Operation was declined by the family, and two days later the child was absolutely well. A localized edema would seem to be the only possible explanation of such an occurrence.

Dr. C. A. Herter, of New York—I believe that local effusion can give rise to these symptoms. I have observed a case of extreme general edema of the arachnoid following syphilis. There was some doubt as to whether the fluid was a simple effusion or an exudate; the inflammatory process, however, could hardly occur so rapidly. The symptoms may possibly be due to cerebral anemia, dependent on compression of the brain by the fluid. Pronounced general edema of the brain in children may be accompanied by any cerebral symptoms.

Dr. Joseph Collins reported the following case as an example of post-traumatic, circumscribed, serous meningitis, with paralytic symptoms due to pressure:

A man, about fifty years old, was admitted to the City Hospital in the wards of Dr. Brewer, with whom I saw him. His history, which was obtained from Gouveneur Hospital, to which he had been taken immediately after the accident, was that he had fallen from a hay-loft; in falling had struck his head, and had received an open wound of the scalp over the left temperoparietal region. This, aside from a moderate degree of shock, seemed to be the only effect of the accident. The wound was dressed, and the patient received otherwise no special attention. He soon became stupid, slightly irrational, and complained, at times, of headache. He was transferred to the City Hospital, after the scalp wound had healed, and examination at this time showed:

1. Mental sluggishness; slowness in answering questions—frequently the patient would stop in the middle of an answer and not finish it; a dazed, non-attentive, dreamy, uncommunicative state; an absence of demands of any kind, but response to the calls of nature.

2. Paralysis of the right upper extremity, principally of the shoulder, reminding one very much of a "deltoid" paralysis; the possibility of movement of the fingers and hand, and of flexion and extension of the forearm in a limited degree.

3. Temperature slightly elevated; respirations sometimes slow, sometimes rather accelerated.

4. Slight stiffness of the neck in varying degree.

5. Pupils of moderate size, equal and of slight response either to light or in accommodation. It is probable that they did not react in accommodation.

6. Absence of cranial nerve involvement, so far as could be made out.

7. Inability to stand or walk.

8. Knee jerks elicitable, but both very weak, and the right more so than the left.

9. Necessity of catheterization. Later there was some dribbling.

The symptoms became aggravated; the mental obtundity more profound; the paralysis of the right upper extremity more complete; the stiffness of the neck less marked; and the impairment of vital functions more evident. The patient was unable to make any response, and the paralysis extended to the right leg.

A diagnosis of intrapial hemorrhage or edema, situated over the lower end of the Rolandic region of the left hemisphere, was made, and the surgeon was advised to operate over the shoulder centre of the left hemisphere. The operation was undertaken, and on removal of a flap of bone from the skull over the left Rolandic region, the dura was ruptured, and about three ounces of clear fluid gushed out. On examination it was seen that the pia was adherent to the dura and the dura slightly adherent to the cranium, but sufficiently to cause rupture when the bone flap was pulled back. After the fluid had escaped there seemed to be a profound concavity in the surface of the hemisphere corresponding to the seat of the exudation. The finger introduced into the skull opening, and passed along the parietal pia, failed to detect any further abnormality, and the wound was closed.

The patient withstood the operation very well, but there was no improvement in his general condition, and he died five days later.

The autopsical findings were unlike anything previously encountered. The only departure from normal within the cranial cavity, with the exception of a comparatively slight degree of ventricular distension, was the deep circumscribed concavity of the lower Rolandic area of the left hemisphere already spoken of. It was impossible to find the reason for the limitation of the cavity, as no membrane could be found. The limiting membrane had been broken down, probably at the time the surgeon swept his finger around the concavity in search of further abnormalities. The gyri were flattened and sunken in, and the fissures were smoothed out in this concave area, and on section through the gyri involved the cortex was seen to be very much thinned. The meninges over the rest of the brain were quite normal in appearance. Microscopical sections have yet to be made.

The pathogenesis of this localized, circumscribed, parietal, pial edema is probably the following: After the injury a low grade of serous meningitis developed, and at the borders of this area of meningitis a slight amount of plastic exudate was formed, which was sufficient to unite the two layers of the pia and to give the exudation definite limitation.. This limiting wall was not found on necropsical examination, because in removing the dura, to which in all likelihood the pia was somewhat adherent, it was torn away or broken down, and it is not improbable that future microscopical examination of the pia will show the remains of this limiting wall.

The absence of indications of irritation referable to the motor area in the beginning of the patient's disorder, and the presence of meningeal symptoms, particularly the psychical ones, had much to do in deciding the diagnosis and the decision of advisability of operation.

In response to an inquiry, Dr. Walton stated that the temperature in these cases is generally only a moderately elevated one, as high as 102 degrees; such as is not always observed in simple hemorrhage.

Dr. J. J. Putnam—I saw an extremely interesting case some years ago, of localized hemorrhage from the veins of the pia mater, where a dense, thick clot eventually formed, causing symptoms, first of irritation, finally of paralysis and coma. If such a clot can be confined without the production of inflammation, within comparatively close limits; I do not see why the same thing might not be true of edema.

The President—The condition of localized edema should not be a question of theory, but a question of fact, and I think that we have testimony enough before us to-day to warrant the conclusion that such a thing is possible—indeed, not only possible, but frequent. I have seen this condition in three cases that I recall where operations have been undertaken, and where a distinct condition of subarachnoid edema, not in any respect inflammatory, was present, and subsided—could be watched in its subsidence—during the operation; returning subsequently to the operation—in one case two days after the operation—and then was again relieved under dressing; each time, at the period of the return, producing definite symptoms. So that in the living subject I believe we have proof of localized edema of the arachnoid and localized edema of the brain.

In a post-mortem examination recently made in connection with Dr. Peterson in a case of brain tumor, where the tumor was not of large size, both Dr. Peterson and myself were impressed by the large amount of edema of the entire hemisphere in which the tumor lay. The edema was so great as to increase the measurements of the hemisphere certainly by an inch, and yet the tumor itself was very small, there being therefore a very distinct edema of very marked character around the tumor. Nervous pathology should come into line with pathology in other parts of the body, and there is no reason why edema should not occur in connection with tumors and small lesions in the brain as well as in other parts of the body. Dr. Janeway, speaking of this particular case, called to mind two cases in which he had seen coma followed by apparent recovery, and then coma and death, in which the only explanation of such alternation in the symptoms in a condition of brain tumor, where the tumor was found at the time of autopsy, was to be found in the existence of a transient edema, which had disappeared and then returned. I think this discussion has been of considerable interest, and we are indebted to Dr. Walton for bringing the subject before us.